Techniques and formulations for administering the compositions comprising iron hemoprotein generally may be found in *Remington's Pharmaceutical Sciences,* Meade Publishing Col., Easton, Pa., latest edition. In a preferred embodiment, the hemoglobin is administered intravascularly. The amount of iron hemoprotein administered, the effective amount, is an amount sufficient to bind to substantially all of the nitric oxide present, thus removing free nitric oxide from circulation. Such an effective amount is generally in a range of from about 0.1 g/kg body weight to about 10 g/kg body weight.

The following examples are presented to illustrate a best mode and preferred embodiments of the present invention and are not meant to limit the claimed invention unless otherwise specified.

EXAMPLE I

Abrogation of Blood Pressure Decline

Experiments were carried out on conditioned mongrel dogs weighing 23–28 kg. Animal care was in accordance with the recommendations of the American Association for Accreditation of Laboratory Animal Care and met all standards prescribed by the *Guide for the Care and Use of Laboratory Animals* (Committee on Care and Use of Laboratory Animals (1978) *Guide for the Care and Use of Laboratory Animals* (Natl. Inst. Health, Bethesda, Md.) DHEW Publ. No. (NIH) 78–83.

The dog was fasted overnight before the day of the experiment and was anesthetized with pentobarbital (25 mg/kg, i.v.), orotracheally intubated and ventilated with a Harvard pump at a nominal rate of 12 breaths per minute and a tidal volume of 15 ml/kg. The dog was instrumented with a cordis for the introduction of a Swan-ganz catheter for measurement of central venous and pulmonary arterial pressures as well as an arterial line for continuous blood pressure monitoring via a computer driven analog to digital processor.

After the blood pressure and heart rate was stabilized, endotoxin was administered to the dog as a bolus injection (50 μg/kg, i.v. in 10 ml DPBS. After the onset of hypotension, a rapid infusion of 50 ml hemoglobin (5.0 g commercially available bovine hemoglobin/100 ml) over 20 seconds was administered by a central venous catheter. Cardiovascular changes were monitored for an additional 12 hrs. after hemoglobin administration.

A tracing depicting endotoxin-induced hypotension, its inhibition by hemoglobin, and subsequent administration of bovine serum albumin (5 grams) is shown in FIG. **1**. Mean arterial blood pressure declined from 160 mm Hg to 70 mm Hg two hours after endotoxin administration. The hemoglobin (HgB) was subsequently administered as an intravenous bolus. The blood pressure drop was abrogated by this administration and the hypotension was partially reversed. This effect lasted approximately 45 minutes before the blood pressure decline resumed. The subsequent decline was only slightly influenced by the administration of 5 grams of bovine serum albumin (BSA). On repetition of this experiment, substantially the same results were obtained.

EXAMPLE 2

Binding of NO by Myoglobin

Nitric oxide formation by endothelial cell cytosol was measured by a novel kinetic 96-well microplate assay. The assay is based on the capture of NO by Fe^{2+}-myoglobin (Mb) which is subsequently oxidized to Fe^{3+}-myoglobin. The process of heme oxidation was continuously measured in a kinetic microplate reader (Molecular Devices, Menlo Park Calif.) as the rate of change in OD_{405}–OD_{650}. Data points were collected from all 96 wells every 16 sec. for 30 minutes at 250° C. with shaking prior to each OD measurement. The slope of the best fit regression line (OD/min) was used to calculate the rate of NO-synthesis. OD/min measurements were converted to pmoles NO/min by dividing by the increase in OD_{405}–OD_{650} measured upon complete oxidation of 1 pmole of reduced Mb with a 10-fold molar excess of potassium ferricyanide. Under the conditions employed, the efficiency of NO capture by Mb approached 100%; neither doubling nor halving the concentration of Mb influenced the Mb oxidation rate. All samples contained 5–30 μl of endothelial cell cytosol (1–6 mg protein/ml) and final concentrations of 5 μM Mb, 500 μM L-arginine, 500 μM NADPH and 80 Mm TRIS, pH 7.6.

Fe^{2+}-Myoglobin Preparation: 2 Mm myoglobin (from horse skeletal muscle, sigma) was reduced with a sodium dithionite and immediately applied to a Sephadex G-25 column, followed by elution with 50 Mm TRIS buffer, Ph 7.6. Mb was aliquoted and stored at −700° C. for up to 2 months prior to use. The usefulness of this iron hemoprotein for nitric acid removal is thus clearly shown.

Various references are cited above, the disclosures of which are incorporated in pertinent part by reference herein for the reasons cited.

The invention described and claimed herein is not to be limited in scope by the specific embodiments disclosed, since these embodiments are intended as illustrations of several aspects of the invention. Any equivalent embodiments are intended to be within the scope of this invention. Indeed various modifications of the invention in addition to those shown and described herein will become apparent to those skilled in the art from the foregoing description. Such modifications are also intended to fall within the scope of the appended claims.

What is claimed is:

1. A method for alleviating deleterious nitric-oxide induced effects in an animal, the method comprising administering a therapeutically effective amount of an iron hemoprotein to said animal.

2. A method of reducing nitric oxide levels induced in an animal by a cytokine or by endotoxin, the method comprising administering to said animal an iron hemoprotein in an amount effective to bind or oxidize nitric oxide.

3. A method for treatment of systemic hypotension in a patient induced by chemotherapeutic treatment with a cytokine, the method comprising administering a therapeutically effective amount of an iron hemoprotein to said patient.

4. A method for treatment of systemic hypotension in a septic patient caused by endotoxin-induced nitric oxide production comprising intravascularly administering an amount of an iron hemoprotein sufficient to bind or oxidize substantially all of the nitric oxide produced.

5. The method of claim **1**, **2**, **3** or **4** where the iron hemoprotein is hemoglobin or myoglobin.

6. The method of claim **1**, **2**, **3** or **4** where the amount is from 0.1 to 10 g/kg body weight.

7. The method of claim **1**, **2**, or **3** wherein the administering is intravascular.

8. The method of claim **1** where the deleterious nitric oxide-induced effect is systemic hypotension.